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THE RELATIONSHIP BETWEEN AMBIENT CARBON MONOXIDE LEVELS, POSTMORTEM CARBOXYHEMOGLOBIN, SUDDEN DEATH AND MYOCARDIAL INFARCTION

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THE RELATIONSHIP BETWEEN AMBIENT CARBON MONOXIDE LEVELS, POSTMORTEM CARBOXYHEMOGLOBIN, SUDDEN DEATH AND MYOCARDIAL INFARCTION

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The relationship between carbon monoxide exposure and heart attacks was studied in Baltimore, Maryland. Four hypotheses were tested: (1) Is there a relationship between sudden death and myocardial infarction and ambient carbon monoxide levels, (2) Are postmortem carboxyhemoglobin levels higher for ASHD sudden deaths as compared to sudden deaths from other causes, (3) Are there differences in postmortem carboxyhemoglobin levels between ASHD sudden deaths and living controls, and (4) Is there any difference in the pathologic characteristics of ASHD sudden death with high or low carboxyhemoglobin levels. There was no evidence of clustering of either myocardial infarction or sudden ASHD on a specific day, nor between the number of cases perday and ambient carbon monoxide levels.

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ABSTRACT

The relationship between carbon monoxide exposure and heart attacks was studied in Baltimore, Maryland.

Four hypotheses were tested: (1) Is there a relationship between sudden death and myocardial infarction and ambient carbon monoxide levels, (2) Are postmortem carboxyhemoglobin levels higher for ASHD sudden deaths as compared to sudden deaths from other causes, (3) Are there differences in postmortem carboxyhemoglobin levels between ASHD sudden deaths and living controls, and (4) Is there any difference in the pathologic characteristics of ASHD sudden death with high or low carboxyhemoglobin levels.

There was no evidence of clustering of either myocardial infarction or sudden ASHD on a specific day, nor between the number of cases per day and ambient carbon monoxide levels.

Postmortem carboxyhemoglobin levels were slightly higher for ASHD sudden deaths than for sudden deaths due to other causes. Any differences were probably primarily due to cigarette smoking.

Cigarette smokers who died suddenly due to ASHD had substantially higher postmortem carboxyhemoglobin levels than nonsmokers. Practically all of the elevated carboxyhemoglobin levels could be related to cigarette smoking or specific environmental exposure. There were no differences in carboxyhemoglobin levels between ASHD sudden deaths and living controls. There was also no relationship between cardiac pathology and postmortem carboxyhemoglobin levels among ASHD sudden deaths.

THE RELATIONSHIP BETWEEN AMBIENT CARBON MONOXIDE LEVELS, POSTMORTEM CARBOXYHEMOGLOBIN, SUDDEN DEATH AND MYOCARDIAL INFARCTION

Clinical, experimental and epidemiological studies have shown a relationship between carbon monoxide exposure and various aspects of clinical coronary artery disease.[1]

Major sources of carbon monoxide can be subdivided into three categories:[2] (1) tobacco smoking, (2) occupational, and (3) community air pollution.

Clinical and experimental studies of the relationship between carbon monoxide and clinical coronary artery disease have noted an increase in symptoms of angina among men exposed to heavy freeway traffic in Los Angeles,[3] or after breathing 50 or 100 ppm of carbon monoxide for up to four hours.[4] Acute exposure to high levels of carbon monoxide resulted in increased coronary blood flow in patients without coronary disease, but not in those with coronary disease.[5]

Experimental studies have also shown a possible relationship between carbon monoxide exposure and the development of atherosclerotic-like disease. Rabbits exposed to moderate doses of carbon monoxide (carboxyhemoglobin 11%) were noted to develop focal degenerative and reparative vascular changes which were possibly precursors of atherosclerotic plaque formation.[6] Similarly, exposure of rabbits to carbon monoxide and a high cholesterol diet enhanced the development of atherosclerosis when compared to feedings of cholesterol alone.[7]

Epidemiological studies have clearly shown an increased risk of coronary artery disease among cigarette smokers.[8,9] The increased risk has been noted primarily for sudden death and myocardial infarction, and much less so for angina pectoris.[10] The risk is directly correlated with the number of cigarettes smoked. Furthermore, the risk of coronary disease was substantially decreased following cessation of cigarette smoking.

More recent experimental and epidemiological studies have strengthened the association between atherosclerotic cardiovascular disease, smoking and carbon monoxide. Individuals who smoked nonnicotine cigarettes had increased carboxyhemoglobin levels. Following the smoking of cigarettes, there was a decrease in time that patients with angina could exercise before developing chest pain.[11,12]

Few studies have attempted to correlate ambient carbon monoxide exposure and coronary disease in a community. In Los Angeles, postmortem blood carboxyhemoglobin levels were measured in a sample of deaths certified at the coroner's office.[13] The carboxyhemoglobin levels were higher for cigarette smokers than non-smokers. The postmortem carboxyhemoglobin levels were weakly but positively correlated with the ambient carbon monoxide exposures just prior to death. A further analysis of the same data failed to note any significant difference in

the postmortem carboxyhemoglobin levels between individuals dying of myocardial infarction or other cardiovascular disease.[14]

In the Los Angeles region, the case-fatality percentage for hospitalized myocardial infarction patients was greater in the areas with high ambient carbon monoxide (8 to 14 ppm) than in the lower areas.[15,16] The differences were apparent during the weeks of increased pollution (weekly mean CO of 7.7 to 14.0). In the higher polluted areas there were also positive correlations between the ambient carbon monoxide levels and the daily myocardial infarction case-fatality percentage in the hospital. There was, however, no association between ambient carbon monoxide levels and the number of admissions for myocardial infarction each day.

No other study has attempted to relate the ambient carbon monoxide levels to either the incidence, case-fatality or deaths due to ASHD. An ongoing study of sudden death and myocardial infarction in Baltimore City made it possible to relate the incidences of myocardial infarction and sudden death to ambient carbon monoxide levels and blood carboxyhemoglobin. Since approximately 70% of all ASHD deaths occur outside of a hospital and approximately two-thirds of the ASHD deaths were sudden, the Baltimore study included both hospital and non-hospital ASHD deaths, with a particular emphasis on ASHD sudden deaths since their time of onset could be more clearly identified.

Four specific hypotheses were tested:

1. Is there a temporal relationship between the incidence of sudden cardiac death, transmural myocardial infarction or total ASHD deaths in a community and the ambient carbon monoxide level?

2. Are the postmortem carboxyhemoglobin levels higher for ASHD sudden deaths as compared to sudden deaths from other causes?

3. Are there differences in the postmortem carboxyhemoglobin levels between ASHD sudden deaths and normal living controls after adjustment for differences in cigarette smoking?

4. Are there differences in the pathology of sudden cardiac death in relation to carboxyhemoglobin levels or cigarette smoking?

METHODS AND RESULTS

1. Relationship Between Events Per Day and Ambient Carbon Monoxide Levels:

The first hypothesis of a relationship between events per day and ambient carbon monoxide levels was tested in the following ways. All sudden, non-traumatic deaths, patients hospitalized with a transmural myocardial infarction and non-sudden

deaths due to arteriosclerotic heart disease, between the ages of 25-64, were identified during a two-year period, June 1970-June 1972, in a defined geographic area of Baltimore City. This area had a population of about 500,000. The detailed surveillance methods have been previously described.[17] During the two year period, 522 ASHD sudden deaths, 336 ASHD non-sudden deaths and 539 transmural myocardial infarctions were ascertained.

The methods of case ascertainment have been described previously.[17] Sudden death was defined as occurring within 24 hours of onset to individuals able to function without assistance outside of their home. The deaths were further subclassified by length of survival, history of heart disease and by whether witnessed or not. Cause of death was based on a review of history, clinical records and postmortem examinations. Approximately 50% of the ASHD sudden deaths were certified by the medical examiner. For the sudden deaths certified by the medical examiner, a detailed study of the pathology of sudden nontraumatic deaths was conducted. About 95% of the sudden deaths certified by the medical examiner had a postmortem examination, including a special examination of the heart to assay the degree of coronary stenosis, presence of acute lesions, and types of myocardial pathology.

Transmural myocardial infarction was defined as admission for a heart attack in which there were evolving "Q" and "ST-T" wave changes on the electrocardiogram with or without serum enzyme changes. All cases were reviewed independent of the ambient carbon monoxide levels.

During approximately a concurrent nine-month period of the surveillance study described above (November 1, 1971-August 31, 1972), a special air pollution monitoring station was operated at the Johns Hopkins School of Hygiene and Public Health. Ambient carbon monoxide levels and other pollutants were measured continuously. The number of new sudden, non-sudden ASHD deaths and living myocardial infarction patients occurring on each day was determined and compared with the expected number of cases per day, assuming a random poisson distribution.

There was no evidence of any clustering of sudden deaths, myocardial infarctions or non-sudden deaths on any specific day within the geographic area of the surveillance study (Figure 1). The absence of any clustering of events over time would be evidence against a major effect of an environmental factor, presuming the level of the environmental factors varied over time.

The distribution of cases in the defined area was then compared with the 24 hour mean ambient carbon monoxide levels measured at the Hopkins station (Table 1). There was no evidence of any association between the ambient carbon monoxide levels and the number of cases of any type per day. Because of the relatively

small number of days with high ambient carbon monoxide levels, the analysis was continued throughout the summer of 1972.

There was still no evidence of any association between the carbon monoxide levels and number of cases. The only time there was any evidence of a relationship between an environmental factor and clustering of events occurred in July 1972 when during a brief "heatwave" on July 24-25, there were six deaths on July 24 and six on July 25 in the surveillance area. The ambient carbon monoxide level remained low during this period. Therefore, the surveillance mechanisms were able to identify a possible environmental precipitent such as excessive temperatures. However, the first hypothesis that ambient carbon monoxide was correlated with the distribution of heart attacks was unsupported as we found no evidence of clustering of cases in time or relationship between number of cases per day and the ambient carbon monoxide level.

As previously noted, the Los Angeles Study included both a spatial and temporal distribution in relation to ambient carbon monoxide levels. The spatial distribution of events in relation to ambient carbon monoxide in Baltimore could not be analyzed for two reasons:

a. Approximately 70% of the ASHD deaths were sudden and occurred outside of a hospital. It would be necessary to pinpoint the area of onset for each event and relate it to a "carbon monoxide level." Unfortunately, there would have been no way of obtaining a denominator, the number of individuals at risk in that specific area at the time of the event.

b. In order to compare events in each area, we needed to have measurements of ambient carbon monoxide levels within specific subareas. Unfortunately, the number and quality of the sampling stations precluded any detailed subarea analysis.

2. Relationship between Postmortem Carboxyhemoglobin and Cause of Death

In order to determine the relationship between postmortem carboxyhemoglobin levels and causes of sudden death, postmortem blood specimens were collected from 2,366 deaths certified by the medical examiner of the State of Maryland. This sample included deaths in the Baltimore City surveillance area and also from the rest of the City and State of Maryland. All age groups and both traumatic and non-traumatic sudden deaths were included.

The Carboxyhemoglobin levels were measured in duplicate by a spectophotometric technique that determined both carboxyhemoglobin and methemoglobin at the same time.[18] The technique was carefully validated for postmortem blood specimens of

carboxyhemoglobin by comparison with gas chromatography. Carboxyhemoglobin levels remained stable after death.[19] This observation has also been reaffirmed in the present study.

The cause of death for these 2,366 people and circumstances associated with the deaths were ascertained from the death certificates and medical examiner's reports, including an autopsy examination in a high percentage of the deaths.

The carboxyhemoglobin levels were slightly higher for the ASHD sudden deaths than for sudden deaths from other natural causes and lower than for deaths due to homicides (Table 2, Appendix Table 1). The differences were small and can probably be explained by variations in smoking habits. Unfortunately, data on smoking habits were obtained for too few of the sudden deaths from other causes than ASHD, so this hypothesis could not be critically tested.

In a recent attempt to study the association between carboxyhemoglobin, smoking and causes of death, blood thiocyanate and carboxyhemoglobin levels were measured. Thyocyanate levels are a measure of long-term smoking experiences. Preliminary results showed that the differences in carboxyhemoglobin levels among the cause of death groups were primarily a reflection of the recency of cigarette smoking prior to death.[20]

Since we had previously noted that cigarette smokers had carboxyhemoglobin levels as high as 10% or more, the characteristics of death with high carboxyhemoglobin levels greater than 10% were studied. We presumed that at least some of these higher levels were not due to cigarette smoking alone. Note in Table 3 that many of these deaths were related to fires or suicide by carbon monoxide. Only 1% of the ASHD deaths had carboxyhemoglobin levels that were greater than 10. The circumstances associated with these ASHD deaths will be discussed later in this report.

Among the ASHD sudden deaths, carboxyhemoglobin levels were higher in the younger as compared to the older age groups (Table 4). Seven percent of the 846 ASHD sudden deaths had a postmortem carboxyhemoglobin level greater than 4% (Table 4). Above the age of 65 when it is believed that individuals are most susceptible to air pollution effects, only 6 of 341 deaths had levels greater than 4% and over 75% were below 1%.

The relationship between the postmortem carboxyhemoglobin levels for ASHD sudden deaths and living controls was determined as follows:

For the ASHD sudden deaths within the special surveillance study area in Baltimore City, a detailed follow-back interview was completed with the next of kin. Information about place of death, activity at onset, length of survival, smoking history and prior history of disease was collected. Interview information could be collected

for about 90% of the ASHD sudden deaths. The data were collected independent of the carboxyhemoglobin levels and the results of the postmortem examination.

Smoking histories and postmortem carboxyhemoglobin levels were reported in 243 ASHD sudden deaths. This included 120 deaths in which there was a special detailed postmortem examination. Postmortem carboxyhemoglobin levels were substantially higher for the cigarette smokers than the nonsmokers (Table 5). Note that 86% of the nonsmokers had carboxyhemoglobin levels less than 2%.

An attempt was then made to evaluate the circumstances at the time of death for cases with high levels of carboxyhemoglobin. Of the eight deaths among nonsmokers with carboxyhemoglobin levels greater than 3, seven were former smokers and three also had possible specific environmental exposures (Table 6). It is certainly possible that some of the current nonsmokers were still smoking prior to death since the smoking histories were obtained from the next of kin and other relatives rather than from the deceased. Of the seven who were current smokers and had levels greater than 10% carboxyhemoglobin, four had a definite environmental exposure beside heavy cigarette smoking and one was a heavy smoker with chronic emphysema.

3. Comparison of Carboxyhemoglobin between ASHD Sudden Deaths and Living Controls:

The distribution of carboxyhemoglobin levels among the ASHD sudden deaths and living controls after adjustment for the history of cigarette smoking is shown in Table 7. Living controls who were cigarette smokers had higher carboxyhemoglobin levels than ASHD sudden deaths who had smoked cigarettes prior to death. The sudden deaths with a history of not smoking cigarettes prior to death had higher carboxyhemoglobin levels than the nonsmoking living controls. The nonsmokers included both those who had never smoked cigarettes and ex-smokers.

The most likely explanation for these results was, for the nonsmokers probably at least a few were actually still smoking prior to death, even though their spouse or other relatives thought they were nonsmokers. In order to test this hypothesis we compared the distribution of carboxyhemoglobin levels among individuals who had never smoked cigarettes. As noted in Table 7, there were no differences between the sudden deaths and living controls, and all but two were below 2% carboxyhemoglobin levels. Some of the current cigarette smokers among the sudden ASHD deaths were probably "sick" prior to death and had stopped smoking; hence their lower carboxyhemoglobin levels.

Other characteristics of the ASHD sudden deaths in relation to carboxyhemoglobin levels were also reviewed. There was no substantial difference in postmortem carboxyhemoglobin levels among the ASHD sudden deaths in relation to activity

or place of onset of the heart attack. There was also no difference in postmortem carboxyhemoglobin levels by length of survival or whether the death was witnessed or not.

4. Relationship between Postmortem Carboxyhemoglobin and Pathology of Sudden Deaths:

An attempt was made to include a detailed postmortem examination for every ASHD sudden death certified by the medical examiner within the special geographic surveillance area in Baltimore City. Of the 522 ASHD sudden deaths, 258 were certified by the medical examiner and 231 of the 258 had a postmortem examination. Approximately 85% (171) of the 231 had a special detailed examination of the heart in order to ascertain the extent of coronary artery stenosis, prevalence of acute lesions in the arteries and myocardial pathology. The special postmortem examination study began prior to the analysis of the carboxyhemoglobin levels so, only 120 of the 171 had both a detailed postmortem examination and carboxyhemoglobin determination. The distribution of postmortem carboxyhemoglobin levels was similar among the deaths with an acute pathological lesion such as recent coronary thrombosis or hemorrhage in a plaque, and those without acute lesions. There was also no difference in postmortem carboxyhemoglobin levels in relation to the maximum degree of coronary stenosis or the number of arteries with greater than 50% stenosis.

Practically all of the ASHD sudden deaths had severe coronary artery stenosis, 102 of the 120 had three or four coronary arteries (left main, anterior descending, circumflex or right coronary artery) with 50% or greater stenosis. The ASHD sudden deaths therefore represent very severe coronary artery diesease and probably should be most responsive to change in ambient carbon monoxide levels.

DISCUSSION

Exposure to carbon monoxide can have major physiological effects on the coronary circulation.[1] Moderately higher levels of carbon monoxide may precipitate symptoms of angina pectoris among susceptible individuals. This study, however, does not support the hypothesis that ambient carbon monoxide levels in an urban area such as Baltimore were major factors in the onset of sudden death and myocardial infarction.

There are several possible reasons that may explain the failure to note such a relationship. First, the levels of ambient carbon monoxide were too low in Baltimore to have any effect. The levels are obviously lower than in areas of Los Angeles or New York, but were probably comparable to other major metropolitan regions. Certainly, a replication of this type of study should be done in an area with presumably much higher ambient carbon monoxide levels. It should be noted, however, that a pilot study of postmortem carboxyhemoblobin levels in Los Angeles reported results very similar to the current Baltimore study.[17]

Second, the major sources of carbon monoxide are occupational, environmental or personal exposure from cigarette smoking rather than general ambient carbon monoxide exposures. The risk of sudden death is much higher among smokers than nonsmokers. Thus, the highest risk group may not be substantially adversely affected by usual community ambient carbon monoxide levels. Cigarette smokers in areas with high ambient carbon monxide levels do, however, have higher carboxyhemoglobin than smokers from less polluted areas.

Finally, there may be a fairly long time period between carbon monoxide exposure and the sudden death or onset of a myocardial infarction. Thus, carboxyhemoglobin levels measured at the time of death or onset of a myocardial infarction may not reflect the carbon monoxide level that precipitated the sudden death or myocardial infarction. There was, however, no relationship between carboxyhemoglobin levels and length of survival. Many sudden deaths did have previous symptomatology and perhaps the premonitory phase was related to carbon monoxide exposures. Prospective type studies of the relationship between symptoms and carbon monoxide levels might be useful. For example, it would be interesting to determine whether patients who have angina pectoris and are nonsmokers have a greater frequency and intensity of symptoms during days in which the ambient carbon monoxide levels are elevated. Although cigarette smokers in areas with high ambient carbon monoxide levels tend to have higher carboxyhemoglobin levels than those living in less polluted areas, the differences are small in comparison to the variations between smokers and nonsmokers. Since most ASHD deaths are sudden and usually associated with an arrhythmia, it might be possible to determine whether patients with angina pectoris or survivors of a myocardial infarction have

a higher frequency of ventricular arrhythmias when the ambient carbon monoxide levels are high.

If the elevation of carboxyhemoglobin levels following cigarette smoking is a major factor in the higher risks of heart attacks among cigarette smokers, then the potential for the development of a "safe cigarette" is extremely remote and possibly the only type of safe cigarette would be a noncombustible variety.

Certainly, much more emphasis should be given to carbon monoxide as a precipitant of heart attack. Much more emphasis, however, should be aimed primarily at cigarette smoking and occupational and unusual environmental exposures than to general ambient carbon monoxide levels, except in unusual environmental circumstances such as the Los Angeles area.

DISTRIBUTION OF NUMBER OF CASES PER DAY DURING 2-YEARS,
COMPARISON OF OBSERVED WITH EXPECTED CASES:
SUDDEN DEATH DUE TO ARTERIOSCLEROTIC HEART DISEASE -- AGES 25-64

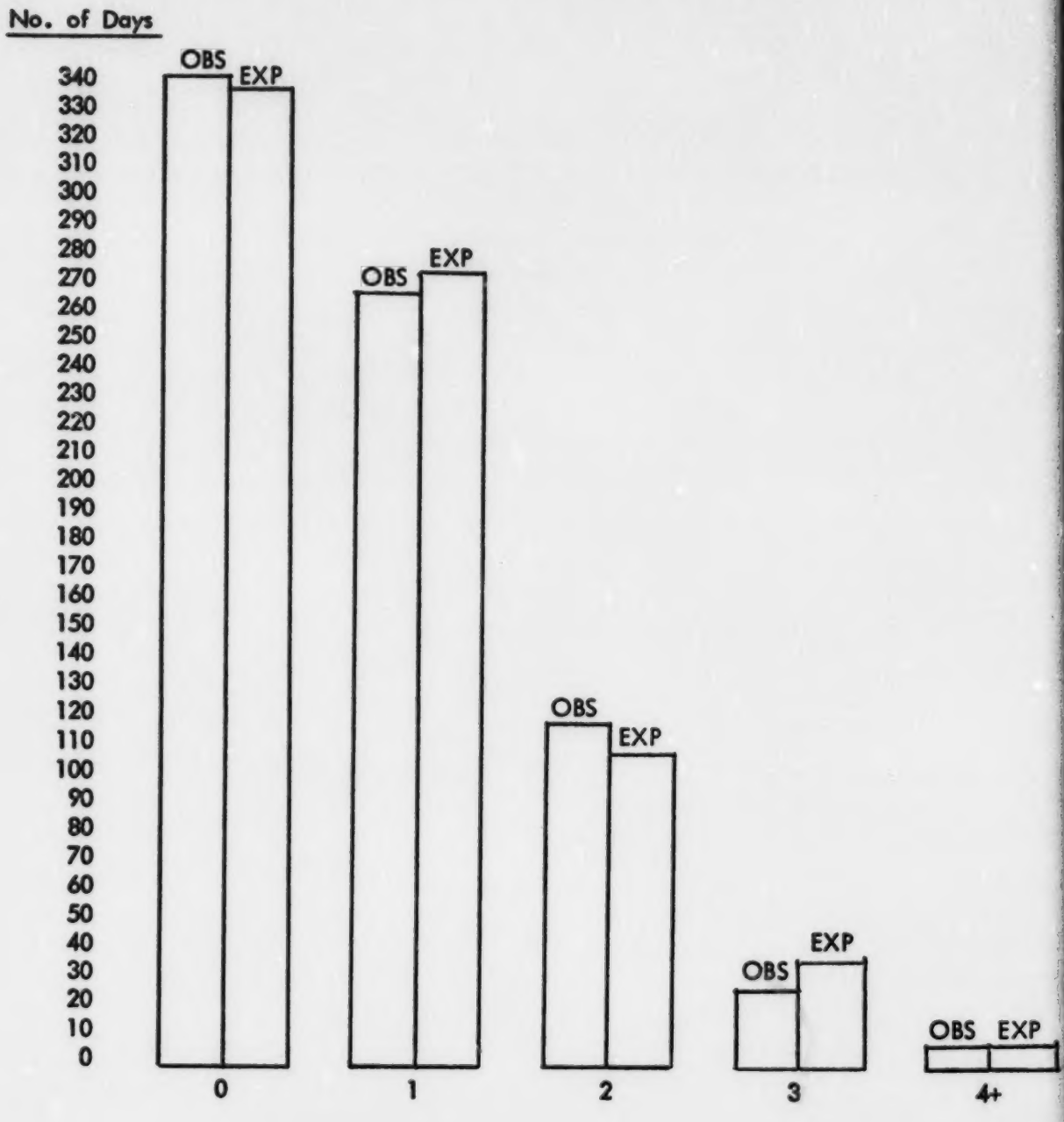

TABLE 1

DISTRIBUTION OF CASES PER DAY IN RELATION TO MEAN CARBON MONOXIDE LEVELS AT HOPKINS STATION

CO AVERAGE 24 Hrs. PPM	TOTAL DAYS	TOTAL CASES* PER DAY IN PERCENT					SUDDEN ASHD DEATHS PER DAY IN PERCENTS				
		0	1	2	3	4+	0	1	2	3	4+
0-4	145	16	33	24	15	12	55	30	13	02	-
5-9	31	13	39	16	22	10	48	32	20	-	-
9+	20	20	15	50	10	05	50	40	10	-	-

*Sudden and Not-Sudden ASHD Deaths and Living M. I. Patients

TABLE 2

COMPARISON OF POSTMORTEM CARBOXYHEMOGLOBIN LEVELS BY CAUSE OF DEATH & AGE

AGE	45-54								55-64							
Cause of Death	ASHD		Other Natural		Auto Accidents		Homicides		ASHD		Other Natural		Auto Accidents		Homicides	
Total	209		169		21		41		225		122		13		27	
Carboxyhemoglobin Level	CUMULATIVE PERCENTS								CUMULATIVE PERCENTS							
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0 - .9	75	36	99	59	11	52	17	41	124	55	89	73	6	47	12	44
1 - 1.9	33	56	37	81	4	71	1	43	34	70	15	85	1	55	5	63
2 - 2.9	27	65	9	86	1	76	3	50	22	80	7	90	3	78	3	74
3 - 3.9	30	79	11	93	2	85	4	60	24	91	9	97	1	86	2	81
4+	44	100	13	100	3	100	16	100	21	100	2	100	2	100	5	100

TABLE 3

Distribution of Postmortem Carboxyhemoglobin Levels by Cause of Death. Levels >10% Carboxyhemoglobin

Cause of Death	No. Deaths	Greater than 10% Carboxyhemoglobin		Percent (Down) (of All Cases >10% Carboxyhemoglobin)
		No.	Percent	
ASHD	852	9	01	11
Other Non-Traumatic	599	-	-	-
Auto Accident	162	2	01	02
Other Accidents	190	9	05	12
Homicide	357	9	03	12
Suicide	52	3	06	04
CO Suicide	24	21	88	27
Fire	34	25	74	32
TOTAL:	2,366	78	03	100

TABLE 4

Cumulative Percent Distribution of Postmortem Carboxyhemoglobin Levels by Age: ASHD Deaths

Age	25-34		35-44		45-54		55-64		65-74		75+		Total	
Number	17		55		209		224		202		139		846	
Carboxyhemoglobin Levels	CUMULATIVE PERCENTS													
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0 - .9	6	35	19	35	75	36	124	55	147	73	112	81	483	57
1 - 1.9	6	70	10	53	33	56	34	70	32	89	16	93	131	72
2 - 2.9	1	76	4	60	27	65	22	80	11	94	9	99	74	81
3 - 3.9	1	82	10	78	30	79	23	91	6	97	-	-	70	89
4 - 4.9	-	-	6	89	19	88	5	93	3	98	1	99	34	93
5 - 5.9	2	94	2	93	8	92	5	95	1	98	1	100	19	95
6 - 6.9	1	100	1	95	9	96	4	97	-	-	-	-	15	97
7 +	-	-	3	100	8	100	7	100	2	100	-	-	20	100

TABLE 5

Distribution of Postmortem Carboxyhemoglobin Levels Among ASHD Deaths By Cigarette Smoking History: (Current Smoking Habits)* Cumulative Percent of Carboxyhemoglobin

	Cigarettes Only	Nonsmokers
NUMBER OF PEOPLE	163	58
Carboxyhemoglobin Level in Percent	CUMULATIVE PERCENT	
0 - 0.9	13	43
1 - 2.9	55	86
3 - 4.9	82	91
5 - 6.9	90	96
7 - 9.9	95	98
10+	100	100

*Excludes pipe and cigar smokers.

TABLE 6

Reasons for High Carboxyhemoglobin Levels Among ASHD Deaths:
By Smoking History

Eight nonsmokers, levels greater than 3	Seven smokers* carboxyhemoglobin level ≥ 10
Seven were former smokers	One had severe emphysema; smoked 2 packs of cigarettes per day
Three also had environmental exposure: Two driving or fixing a car	Two patients smoked 2 packs per day, one of whom was found in a car he was repairing; with the motor running
One found in the basement of a house with a possible deffective heating system, although definite CO exposure was not proven	One found dead in a car
	One died in a home with a defective heating system and definite CO exposure
	Two found dead at home -- no definite environmental factor identified

*Included one cigarette smoker who also smoked a pipe

TABLE 7

Comparison of Carboxyhemoglobin Levels Among ASHD Sudden Deaths and Living Controls By Current Smoking History* (Cumulative Percents)

	Cigarettes				Non-smokers, Inc. Ex-smokers				Excluding Ex-smokers			
	ASHD		Living Controls		ASHD		Living Controls		ASHD		Living Controls	
Carboxyhemoglobin Level in Percents	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0 - 0.9	25	14	7	06	28	48	73	70	12	57	34	64
1 - 2.9	71	54	30	35	22	86	31	99	8	95	18	98
3 - 4.9	50	82	31	64	3	91	1	100	1	100	1	100
5 - 6.9	16	91	26	88	3	96	-	100	-	-	-	-
7 +	16	100	13	100	2	100	-	-	-	-	-	-
TOTAL	178		107		58		105		21		53	

*Smoking history prior to death or at time of interview

APPENDIX TABLE 1

Median and Mean Carboxyhemoglobin Levels by Age and Cause of Death

Cause of Death	AGE							
	25-34		35-44		45-54		55-64	
	Median	Mean	Median	Mean	Median	Mean	Median	Mean
ASHD	1.5	2.0	1.6	2.6	1.9	2.6	0.7	1.6
Other Natural	1.2	1.6	0.8	1.5	0.7	1.3	0.6	1.0
Auto Accident	2.0	3.2	2.3	2.2	0.4	1.8	1.2	2.2
Other Accidents	1.0	5.4	1.2	3.9	1.4	7.1	0.5	1.4
Homicide	2.4	2.9	2.0	2.7	2.9	3.7	1.2	2.1

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